How to recover from alcohol:

* Seek professional medical help if you are experiencing severe withdrawal symptoms.
* Attend a support group, such as Alcoholics Anonymous.
* Find healthy ways to cope with stress and cravings.
* Avoid triggers that may lead to relapse.
* Be patient and persistent in your recovery journey.

If you are struggling with alcohol addiction, there are many resources available to help you. You can find information and support online at findahelpline.com.

Using the Recovery Kernel Framework

Alcohol Use Disorder · Heavy drinking · Beer/wine/spirits · Alcohol-medication dependence.

⚠ HARM REDUCTION RESOURCE — NOT MEDICAL ADVICE | Crisis: call 000 or text 13 11 14

ALCOHOL WITHDRAWAL CAN BE FATAL. READ SECTION 3 FIRST.

A Note Before You Begin

This guide begins with something that no other Recovery Kernel navigation guide has started with: a medical warning that must be read before proceeding.

Abstinence from alcohol can be fatal. This isn't a metaphor, nor a statistical abstraction. It can cause seizures, cardiac arrhythmias, and a syndrome called delirium tremens, which has a mortality rate of up to 15% without medical treatment. This is not the case with cannabis withdrawal or stimulant withdrawal, or even most opioid withdrawals. This applies specifically and distinctly to alcohol—and also to benzodiazepines, which operate through the same mechanism.

If you are a heavy, daily drinker — meaning you drink to function, drink in the morning, experience shaking or sweating or nausea when you go without alcohol for several hours — you must not stop abruptly without medical supervision. The Recovery Kernel framework in this guide fully applies to your recovery. But the first step is not a supplement stack. It is a phone call to a physician or an urgent care clinic or a detox facility.

For moderate to heavy drinkers without physical dependence — those who drink problematically but do not drink to prevent physical withdrawal — the guide below applies more directly. The distinction is clinical and important. If you are not sure which category you fall into, assume the more cautious one and seek medical assessment.

⚠ IF YOU ARE PHYSICALLY DEPENDENT ON ALCOHOL — STOP AND READ THIS NOW.

- Do you drink in the morning to stop shaking?
- Do you experience sweating, a rapid heartbeat, or nausea when you haven't had alcohol for several hours?
- Have you ever experienced seizures or hallucinations when stopping or reducing alcohol consumption?
- Have you been drinking heavily and daily for weeks, months, or years?

If you answered yes to any of the above: do not stop drinking abruptly. Call your doctor, go to urgent care, or contact an alcohol detox facility before reducing or

stopping. This is not a recommendation. It is a medical necessity. Medically supervised alcohol detox with benzodiazepine tapering is standard of care. It keeps people alive. Please use it.

Once you are medically stable — whether through supervised detox or because you have confirmed you are not in the physical dependence category — the full Recovery Kernel framework below is designed for exactly your biology and your recovery journey.

Section 1 — What Alcohol Did to Your Brain

THE CENTURION — The Most Socially Sanctioned Neurotoxin in Human History.

Alcohol is unique among common substances of abuse in that it does not operate through a single receptor system. It touches nearly every major neurotransmitter system in the brain simultaneously — which is why its withdrawal syndrome is more complex and more dangerous than almost any other substance, and why its recovery requires the most comprehensive biological approach.

Comprendere cosa l'alcol fa realmente, non come un fallimento morale, ma come farmacologia, è il fondamento di tutto ciò che segue.

The Five Systems Alcohol Disrupted.

System	What alcohol did?	Withdrawal and Recovery:
		* **Withdrawal:** This refers to the physical and psychological symptoms that occur when someone stops or reduces their use of a substance they are dependent on. The severity of withdrawal symptoms can vary depending on the substance, the amount used, and individual factors. * **Recovery:** This is an ongoing process of healing and growth that involves regaining control over one's life and well-being after struggling with substance abuse or addiction. Recovery is not just about stopping drug use; it also includes addressing underlying issues, developing healthy coping mechanisms, and building a supportive network. It's important to remember that everyone's experience with withdrawal and recovery is unique. There is no one-size-fits-all approach, and what works for one person may not work for another. If you or someone you know is struggling with substance abuse or addiction, it's essential to seek professional help. A healthcare provider can assess the individual's

		needs and develop a personalized treatment plan that addresses both the physical and psychological aspects of recovery. **Here are some resources that can provide support and guidance:** *** [findahelpline.com] (findahelpline.com)**
GABA (the brake).	Potentiated GABA-A receptors — amplified the brain's primary inhibitory system. Alcohol is a powerful GABA-A agonist. The calmness, the reduced anxiety, the social ease, the sleep — all GABA.	With chronic use, GABA receptors downregulate to compensate. When alcohol is removed, the brake system is depleted while the accelerator runs unchecked. Result: seizure risk, tremors, extreme anxiety, insomnia. This is the medical emergency of alcohol withdrawal.
Glutamate (the accelerator).	Inhibited NMDA glutamate receptors — reduced excitatory signalling. The opposite of what stimulants do. Alcohol quiets the excitatory system.	Chronic alcohol use causes glutamate receptor upregulation — the brain compensates for the inhibition by making more accelerators. Remove the alcohol: GABA is depleted, glutamate is upregulated. The brain runs at dangerously high excitation. Seizures are the extreme expression of this imbalance.
Dopamine (el neurotransmisor de la recompensa).	Indirekt stimulerar dopaminfrisättning i nucleus accumbens – belöningscentret. Detta är en sekundär effekt av GABA/glutamat och förklarar varför alkohol känns bra, inte bara lugnande.	Il sistema dopaminergico si riduce in modo simile agli stimolanti, ma più lentamente. L'anhedonia, la bassa motivazione e la depressione nella riabilitazione dall'alcolismo riflettono l'esaurimento del sistema dopaminergico insieme alla carenza di GABA. Anche il sistema del piacere è disattivato, non solo quello calmante.
Serotonin.	Acutely increases serotonin release — contributing to mood elevation in low doses. Chronic heavy use depletes serotonin system function.	Serotonin deficiency during recovery can cause depression, anxiety, irritability, and sleep disturbances. 5-HTP, 5-MTHF, and diets rich in tryptophan are crucial for rebuilding the serotonin system. This is often misdiagnosed as primary depression when it's actually a deficit occurring during the recovery phase.
Opioid system.	Triggers the release of natural opioids—beta-endorphins—which contribute to the feeling of warmth, social connection, pain relief, and euphoria associated	Depletion of endogenous opioids during recovery can lead to increased emotional pain sensitivity, difficulty with social bonding, and a specific type of longing that is often misinterpreted as a craving for alcohol, when it is actually a craving for the endorphin state that alcohol produced. The

	with alcohol, particularly in individuals who carry the OPRM1 A118G gene variant.	OPRM1 genotype determines naltrexone response.

The Acetaldehyde Problem — Unique to Alcohol.

THE CENTURION — The poison your liver is trying to clear.

Alcohol is metabolized in the liver through two steps: first to acetaldehyde (by the enzyme ADH1B), then to acetate (by the enzyme ALDH2). Acetaldehyde is directly toxic — it damages DNA, proteins, and cell membranes, and is responsible for the flushing, nausea, and rapid heartbeat of the hangover.

This pathway exhibits genetic variations that significantly influence the effects of alcohol and its associated risks.

Variant génétique.	The biology.	What this means for your recovery.
ADH1B fast variant (rs1229984)	Alcohol converts to acetaldehyde very rapidly – the toxic intermediate accumulates faster than ALDH2 can clear it.	Fast ADH1B + slow ALDH2 = a really bad hangover, but it might actually offer some protection against excessive alcohol intake. Fast ADH1B + fast ALDH2 = efficient processing, but this can lead to increased alcohol consumption. Understand your genetic makeup.
ALDH2*2 variant (rs671) — the Asian flush gene.	Reduces or eliminates the enzyme that clears acetaldehyde. Heterozygous (*1/*2): approximately 50% activity, flushing reaction. Homozygous (*2/*2): near-zero activity, extreme toxicity.	Homozygous ALDH2*2 means that drinking alcohol is physiologically dangerous – acetaldehyde builds up to toxic levels with every drink. This variant offers protection against Alcohol Use Disorder (AUD) but doesn't prevent it; some people experience severe discomfort from drinking and still develop AUD, leading to further organ damage.
ALDH2*1/*1 (wild type)	Full acetaldehyde clearance capacity — the most common variant in European populations.	No physiological barrier to alcohol consumption from this pathway. Other genetic vulnerabilities (GABRA2, OPRM1, COMT) determine addiction risk. Liver damage from chronic heavy use still occurs regardless of efficient ALDH2 — quantity consumed determines hepatotoxicity.

What Chronic Alcohol Use Does to Your Body — Beyond the Brain

* **Liver damage: Cirrhosis, fatty liver disease.**
* **Heart problems: Cardiomyopathy, high blood pressure.**
* **Pancreatitis.**
* **Weakened immune system.**
* **Increased risk of certain cancers.**
* **Nerve damage (neuropathy).**
* **Muscle weakness.**
* **Digestive issues.**
* **Malnutrition.**
* **Sexual dysfunction.**

THE CENTURION — The Whole System Was Involved.

Alcohol is not merely a brain drug. Years of heavy drinking produce systemic effects that must be addressed in recovery alongside the neurological reconstruction:

- Liver: Fatty liver → alcoholic hepatitis → cirrhosis is the progression with unchecked heavy use. Even without cirrhosis, liver function is often significantly impaired, affecting the metabolism of every supplement and medication in this guide. Liver function tests (LFTs) should be assessed at the start of recovery.
- Thiamine (B1) depletion: Alcohol blocks thiamine absorption. Severe thiamine deficiency produces Wernicke's encephalopathy — a neurological emergency involving confusion, eye movement abnormalities, and loss of coordination. If you have been a heavy drinker and experience any of these symptoms, go to an emergency room immediately. High-dose thiamine (B1) is the treatment. This is not a supplement protocol situation — it is a medical emergency.
- Deficiência de magnésio: O álcool aumenta drasticamente a excreção urinária de magnésio. A deficiência de magnésio piora a ansiedade, a perturbação do sono, o risco de arritmia cardíaca e o limiar de convulsões — tudo isso já está elevado na abstinência alcoólica. A reposição de magnésio é a intervenção suplementar de maior prioridade na recuperação do alcoolismo.
- Problemas en el intestino y el microbioma: El alcohol daña la barrera intestinal, altera el microbioma y produce permeabilidad intestinal ("intestino permeable") que provoca inflamación sistémica, alteraciones del estado de ánimo y antojos. El eje intestino-cerebro (Protocolo 09 y Centurian Paper 18 en el Compendio principal) es un objetivo central para la reparación.
- Cardiovascular: Chronic heavy drinking can cause cardiomyopathy, arrhythmias (especially atrial fibrillation), and high blood pressure. A cardiovascular assessment should be included as part of the initial medical evaluation during recovery.

- Immunosuppression: Chronic alcohol consumption suppresses immune function. Many individuals in early stages of alcohol recovery experience immunological depletion. Nutrition and sleep are the primary tools for immune system restoration.

Section 2 — Your Genomic Blueprint

Alcohol use disorder has one of the highest heritability estimates of any condition in medicine — approximately 50–60%. This is not destiny. It is biology that can be understood, worked with, and addressed. The Recovery Kernel's genomic framework reveals which specific biological systems made alcohol feel necessary, and which ones most urgently need support in recovery.

The DNA Testing Panel for Alcohol Recovery.

Gene	What it controls.	Key variant.	Alcohol recovery relevance.
GABRA2	GABA-A receptor alpha-2 subunit — inhibitory tone baseline.	rs279858 / rs279845 high-risk haplotype.	THE most important alcohol-specific gene in the Recovery Kernel framework. High-risk variants structurally reduce baseline GABAergic inhibitory tone — the brain's brake system runs at chronically low power. Alcohol powerfully activates this underpowered system, producing relief that is genuinely extraordinary for this genotype. Recovery must build alternative GABA support — magnesium, GABA-supporting practices, stress reduction — as first-line priorities.
OPRM1	Mu-opioid receptor — endorphin response and social bonding reward.	A118G (rs1799971)	A118G carriers experience amplified dopamine release in the nucleus accumbens in response to alcohol — a stronger reward signal than the A/A genotype. This is the strongest predictor of naltrexone response in alcohol use disorder. If you carry A118G, naltrexone is likely to be significantly more effective for you than for the average patient. Bring this to your physician.
COMT	Velocidad de eliminación de químicos relacionados con la dopamina y el estrés.	Val158Met (rs4680)	Slow COMT (Met/Met, Sage constitution) is heavily represented in alcohol use disorder — the chronically anxious brain that cannot quiet itself finds alcohol's GABA potentiation to be the most effective off switch available. Anxiety management is the central recovery strategy for this

			profile. Fast COMT (Warrior) also appears — social drinkers seeking dopaminergic stimulation from alcohol's secondary dopamine effects.
ALDH2	Aldehyde dehydrogenase 2 — acetaldehyde clearance.	rs671 (*1/*1, *1/*2, *2/*2)	Important for understanding both the risk profile and the extent of body damage. Individuals with a *2/*2 homozygous genotype who consumed alcohol despite experiencing significant discomfort may have substantial organ damage caused by acetaldehyde, which standard recovery protocols must address. Liver function testing is mandatory. N-acetyl cysteine and liver support supplements become even more critical in these cases.
ADH1B	Alcohol dehydrogenase — converts alcohol to acetaldehyde.	rs1229984 (variant for fast versus slow metabolism).	Fast ADH1B (His48 allele) produces acetaldehyde faster — conferring some natural deterrence but also more rapid toxic exposure if drinking continues. Slow ADH1B allows higher alcohol consumption before acetaldehyde accumulation — associated with higher AUD risk in some populations.
MTHFR	Metilazione: la base di tutta la sintesi dei neurotrasmettitori.	C677T (rs1801133), A1298C (rs1801131)	Alcohol depletes folate and disrupts methylation actively. In individuals with MTHFR variants, this exacerbates an existing deficiency, leading to significant impairment of neurotransmitter synthesis. Supplementation with 5-MTHF and methylcobalamin is the highest priority and often results in a dramatic improvement in recovery early on for people with this profile.
DRD2 / ANKK1	Densidade de receptores de dopamina D2.	Taq1A (rs1800497)	A1 allele (reduced D2 density) is associated with alcohol use disorder through the Reward Deficiency Syndrome pathway. Alcohol provides dopaminergic relief for the reward-deficient brain. Recovery must build multiple dopamine sources — this profile cannot be expected to find sobriety satisfying without deliberate reward architecture.
SLC6A4 / 5-HTTLPR	Serotonin transporter expression.	Short/short allele.	Short/short carriers have higher anxiety sensitivity and a higher risk of Alcohol Use Disorder (AUD) – particularly for the anxious-drinking

			pattern. Serotonin reconstruction through 5-HTP, 5-MTHF, and tryptophan-rich nutrition addresses the biological vulnerability directly. SSRIs may be appropriate depending on severity – discuss with your physician.
CYP2E1	Cytochrome P450 2E1 — metabolises alcohol in the liver; generates reactive oxygen species.	Several polymorphisms (rs2031920, rs3813867)	High CYP2E1 inducibility increases oxidative stress from alcohol metabolism — worsening liver damage and neuroinflammation. N-acetyl cysteine (NAC) and antioxidant support (glutathione precursors, alpha-lipoic acid) are particularly important for this profile.
CYP2C19 / CYP2D6	Metabolism of many medications used in alcohol recovery.	Poor to ultra-rapid metabolizer variants.	Affects the metabolism of naltrexone (CYP3A4), acamprosate (not CYP metabolized - renal), disulfiram, and many psychiatric medications used in the treatment of co-occurring conditions. Poor metabolizers experience higher drug levels; ultra-rapid metabolizers may get an insufficient effect. Essential for medication optimization.

Fenotipificación sin resultados de pruebas.

The compendium's phenotyping tools apply directly here. Ask yourself:

- Did alcohol primarily calm you — reduce anxiety, quiet the mind, make social situations manageable? → Likely Sage COMT (Met/Met) + GABRA2 high-risk. Anxiety is your primary recovery target.
- Did alcohol make you feel alive, social, confident, and stimulated? → Social-reward pattern, likely OPRM1 A118G + possible Warrior COMT. Endorphin system and dopamine support are priority.
- Is there a family history of alcohol use disorder across multiple generations? → A DRD2 deficit and GABRA2 variants are likely factors. RDS is probably a significant component.
- Did alcohol cause you more physical discomfort (flushing, rapid heartbeat) than others seemed to experience? → Possible ADH1B fast variant or ALDH2 variant. Liver support is a high priority.
- Did antidepressants never quite work for you? Did folate supplements seem to help? → Likely MTHFR involvement. Start 5-MTHF immediately.

Section 3 — The Detox Protocol

⚠ LEES DEZE SECTIE VOOR ANDERE ACTIES ALS U NU DRINKT.

3.1 — Medical Detox: When It Is Required

THE CENTURION — The Line That Cannot Be Crossed Without Medical Support.

Abstinência alcoólica é a única forma comum de abstinência de substâncias em que a seguinte sequência é possível, documentada e ocorre sem aviso: ansiedade → tremor → agitação → convulsão → delírio tremens → morte. Essa sequência pode começar dentro de 6 a 48 horas após a última bebida em uma pessoa fisicamente dependente.

Medical detox is required—not recommended—if you meet any of the following criteria:

- You drink daily and have been doing so for more than 2-3 weeks.
- Du erfährst körperliche Entzugserscheinungen zwischen den Getränken: Zittern, Schwitzen, schneller Herzschlag, Übelkeit, Angstzustände, die sich durch Alkohol lindern lassen.
- Have you ever experienced a seizure while withdrawing from alcohol?
- Have you ever experienced delirium tremens – confusion, hallucinations, fever, extreme agitation?
- You are also using benzodiazepines or other GABAergic substances — combined withdrawal is especially dangerous.
- Du har en GABRA2-variant med hög risk – om du vet detta, informera ditt avgiftningsteam.

Medical detox involves managing withdrawal symptoms under medical supervision. This can include:

* **Medications to ease discomfort**
* **Monitoring vital signs**
* **Supportive therapies**

The goal is safe and comfortable transition to a substance-free state. It's not optional, and medical help is not failure.

Detoxificación ambulatoria: adecuada para una dependencia física leve a moderada, con un entorno doméstico seguro y contacto médico diario. Se prescribe una reducción gradual de benzodiazepinas (generalmente

diazepam o clordiazepóxido) para prevenir convulsiones mientras el cerebro se readapta.

Inpatient detox: required for severe dependence, prior seizure history, GABRA2 high-risk profile, co-occurring medical conditions, or absence of a safe home environment. 3–7 days with continuous monitoring.

The CIWA protocol: Clinical Institute Withdrawal Assessment for Alcohol — the standard tool medical teams use to assess and manage withdrawal severity. Ask your detox team about it. Understanding it helps you communicate your symptoms accurately.

⚠ NIET SUPPLEMENTEN VERVANGEN MEDISCHE ONTSLAKING BIJ FYSIEK AFHANKELIJKHEID. SUPPLEMENTEN VOORKOMEN GEEN ONTGINNINGSKRAMPEN.

Il protocollo di integratori e peptidi in questa guida inizia dopo la stabilizzazione medica: non al posto della stessa. Se non si è fisicamente dipendenti, o una volta completata la disintossicazione medica, il quadro di ricostruzione biologica sottostante si applica pienamente.

3.2 — The Withdrawal Timeline: Post-Medically Supervised Detox

THE CENTURION — What the Brain Is Moving Through.

Timeline.	Biology.	Symptoms	What to do.
Hours 6–24 (if not medically supervised — seek help).	GABA systems failing as alcohol clears; glutamate systems surging unchecked.	Tremors, sweating, anxiety, rapid heartbeat, nausea, insomnia.	THIS IS THE MEDICAL WINDOW. Call a doctor, go to urgent care, or call 000. Do not manage this alone if you are physically dependent.
Hours 24–72 (peak medical risk)	Maximum GABA-glutamate imbalance; cortisol surging; autonomic nervous system dysregulated.	Severe anxiety, confusion, possible hallucinations, seizure risk.	Medical supervision required for physically dependent individuals. If medically supervised: benzodiazepine protocol to manage the GABA deficit. Begin high-dose thiamine (B1) immediately — preventing Wernicke's encephalopathy is urgent.
Days 3–7 (stabilization)	Acute medical risk passing; GABA systems beginning to rebound;	Profound fatigue, emotional rawness, sleep disruption, depression,	Begin supplement stack (Section 4). Sleep is the priority — the recovering brain desperately needs it.

	glutamate calming; but significant neurochemical instability remains.	cognitive fog, strong cravings.	Begin nutrition protocol — the depleted system needs rebuilding material. Allow the fatigue.
Weeks 1–4 (The Rebuilding)	Densité des récepteurs GABA en voie de récupération ; normalisation du glutamate ; les systèmes dopaminergiques et sérotoninergiques commencent à se réparer ; le foie commence à retrouver ses fonctions.	Intermittent anxiety, depression, sleep difficulty, mood volatility, cravings that come in waves rather than continuously.	Full supplement protocol in progress. Exercise starts – BDNF is the repair signal. PAWS episodes are possible – see Section 3.3. Start the protocol navigation (Section 7).
Months 1–6	Significant GABA receptor recovery; serotonin and dopamine reconstruction; liver healing if drinking has stopped; brain volume beginning to increase (alcohol causes measurable brain atrophy that reverses in sobriety).	Progressive improvement with episodic difficulty; cognitive clarity returning; emotional range widening; sleep normalizing; physical health measurably improving.	When sobriety begins to feel like a positive state, rather than just the absence of something. The brain is rebuilding itself, and this can be seen through MRI scans. The effort put in now will determine the quality of the next ten years.
Months 6–24+.	Volumen cerebral significativamente restaurado; sistemas de GABA y glutamato normalizados; niveles de dopamina y serotonina recuperados; salud hepática en gran medida restaurada en la mayoría de los casos, sin cirrosis.	Near-normal emotional regulation, cognition, and pleasure; episodic cravings primarily contextual; genuine enjoyment of sober life accessible.	Il cervello, dopo 12-24 mesi di astinenza, mostra una misurazione reversibile della perdita di materia grigia causata dal consumo cronico di alcol. Questo è uno degli argomenti biologici più convincenti a favore della sobrietà prolungata.

3.3 — Post-Acute Withdrawal Syndrome in Alcohol Recovery.

⚠ ATTACKS OF WITHDRAWAL SYMPTOMS (PAWS) CAN LAST FOR UP TO 2 YEARS. THIS IS NOT A RELAPSE. IT IS DUE TO CHANGES IN THE BRAIN'S FUNCTIONING.

Il post-astinenza prolungato nell'alcolismo può essere particolarmente lungo e grave. I sistemi GABA e glutammato, che erano cronicamente sbilanciati, non si normalizzano semplicemente e rimangono normalizzati: oscillano durante il periodo di recupero, causando episodi di ansia acuta, depressione, insonnia e desiderio che possono sembrare indistinguibili dalla fase iniziale dell'astinenza.

PAWS is most commonly triggered by: stress, poor sleep, illness, grief, conflict, high-sugar food, caffeine, and exposure to alcohol cues. The GABRA2 high-risk genotype is associated with more severe and prolonged PAWS.

When a PAWS episode occurs: return to Protocol 01 (Shelter) in the main Compendium. Increase magnesium intake. Contact your support network. Do not interpret the episode as evidence that recovery is not working. It is the nervous system adjusting, not failing.

Section 4 — Biological Reconstruction

This section addresses the simultaneous reconstruction of five systems that alcohol disrupted: GABA/glutamate balance, dopamine, serotonin, the opioid/endorphin system, and the methylation foundation that underlies all neurotransmitter synthesis. These are not sequential targets — they are addressed in parallel, with priorities determined by your specific genomic profile and clinical presentation.

Discuss with your physician or pharmacist before starting, especially given the liver involvement common in alcohol recovery. Some supplements are contraindicated in liver disease.

4.1 — The Emergency Nutritional Replenishment

THE CENTURION — The body was starved while drinking continued.

L'uso cronico e pesante di alcol provoca una specifica crisi nutrizionale che deve essere affrontata prima che qualsiasi altro integratore nel programma di recupero possa funzionare efficacemente. Il cervello impoverito non può sintetizzare i neurotrasmettitori senza precursori. I precursori non possono essere convertiti senza cofattori. I cofattori sono stati sistematicamente distrutti da anni di consumo di alcol.

Nutrient.	Priority level	Target dose.	This information is not available.
Thiamine (Vitamin B1)	CRITICAL — Day 1	100–500mg daily.	Wernicke's encephalopathy prevention. Alcohol blocks thiamine absorption — severe deficiency produces irreversible neurological damage. If any confusion, memory problems, or eye movement abnormalities are present, emergency high-dose IV thiamine is required — go to an ER. For all others: oral thiamine is the highest-priority supplement on Day 1 of recovery.
Magnesium glycinate.	CRITICAL — Day 1	400–600mg elemental daily.	L'alcol aumenta notevolmente la perdita di magnesio nelle urine. La carenza di magnesio peggiora la soglia delle convulsioni, il rischio di aritmie cardiache, l'ansia e l'insonnia, tutti fattori già elevati durante la riabilitazione dall'alcolismo. La forma glicinato offre il miglior assorbimento ed effetto neurologico. Dividere la dose

			tra mattina e sera.
B-Complex (fully methylated).	High — Day 1	B-kompleks med metylerte former.	Alcohol depletes the entire B-vitamin spectrum: B1, B2, B3, B5, B6, B9, B12. Use a B-complex that includes methylated forms (methylcobalamin, methylfolate) — not cyanocobalamin or folic acid. The depleted methylation system cannot convert the inactive forms.
5-MTHF (L-methylfolate)	High — Day 1	400–1000 mcg daily.	Alcohol specifically depletes folate, and standard folic acid supplementation does not address MTHFR-variant methylation impairment. 5-MTHF bypasses the MTHFR enzyme entirely and provides activated folate directly to neurotransmitter synthesis pathways. Often produces noticeable mood improvement within 1–2 weeks.
Zinc.	High — Day 1	25–30mg daily with food.	Alcohol reliably depletes zinc. Zinc is required for GABA receptor function, dopamine synthesis, immune function, and wound healing. Alcohol recovery places all of these systems under demand simultaneously. Zinc is the enabling mineral for this multi-system repair.
Vitamin D3 + K2	High — Week 1	3000–5000 IU of vitamin D3 plus 100 mcg of vitamin K2 daily.	Heavy drinkers are almost universally deficient in Vitamin D. D3 regulates gene expression, mood, immune function, and GABA synthesis. K2 ensures D3-mobilized calcium is directed to bones rather than arteries. Take with a fatty meal for absorption.
Vitamin C	Moderate — Week 1	500–2000mg daily, divided.	Potent antioxidant that addresses the oxidative stress of alcohol withdrawal. Also supports collagen synthesis (alcohol damages skin, gut lining, and connective tissue), immune function, and dopamine synthesis (cofactor for dopamine beta-hydroxylase).
Protein and amino acid support.	High — Day 1	0.8–1g per lb body weight daily.	Alcohol suppresses appetite and provides empty calories that crowd out protein. The depleted amino acid pool cannot synthesize neurotransmitters regardless of cofactor availability. Protein is the raw material. Target 30–50g per

			meal. Eggs, fish, chicken, Greek yogurt, legumes. If appetite is suppressed, protein shakes are appropriate.

4.2 — GABA System Restoration

THE CENTURION — Restoring the brake system that had been neglected for years.

The GABA system does not replenish through supplementation in the same way that the dopamine system can be partially supported with precursors. GABA receptor upregulation is a structural process that occurs through abstinence and time. What supplements can do is support the conditions that allow this upregulation to proceed safely and effectively.

Supplement / approach	Dose.	Timing.	Role in GABA recovery.
Magnesium glycinate (repeated)	300–400mg elemental	Morning and evening.	The most important GABA-supporting supplement. Magnesium is an NMDA antagonist — it reduces glutamate excitotoxicity — AND it is required for GABA synthesis AND it supports GABA-A receptor function. It addresses the GABA/glutamate imbalance from both sides simultaneously. There is no better single supplement for the alcohol-withdrawal nervous system.
L-Theanine	200–400mg	As needed, especially PM.	Increases GABA and glycine; promotes alpha-brainwave state. Non-sedating. Does not produce dependence. Provides genuine calming without the addiction liability of benzodiazepines, which is critical in alcohol recovery where benzo substitution is a documented relapse trap.
Taurine	500–2000mg daily.	With meals.	Taurine is a GABA-mimetic amino acid that acts at GABA-A receptors. Alcohol depletes taurine significantly. Taurine supplementation in alcohol recovery has been studied

			with positive effects on withdrawal anxiety and hepatoprotection. A dual-action supplement: calms the nervous system AND protects the liver.
Inositol.	2–12g dagelijks (verhoog de dosis geleidelijk).	Doser som deles opp og tas med mat.	Modulates GABA and serotonin receptor sensitivity. High-dose inositol has evidence for anxiety and OCD—both common in alcohol recovery. Supports the insulin signalling pathways disrupted by alcohol-induced blood sugar dysregulation.
GABA (direct supplement)	250–750mg	Evening.	Controversial: GABA itself may not cross the blood-brain barrier efficiently. However, peripheral GABA receptors and gut GABA activity contribute to calming, and some clinical experience supports its use in anxiety. Phenibut (a GABA derivative) is NOT recommended — it is addictive and substitutes one GABAergic dependence for another.
Lemon balm extract (Melissa officinalis).	300–600mg standardized.	Evening.	GABA transaminase inhibitor — slows GABA breakdown, increasing available GABA. Gentle anxiolytic effect. One of the safer herbal GABA-supporting options without addiction liability. Useful in the weeks 2–8 window when anxiety is the primary challenge.

4.3 — Serotonin and Dopamine Reconstruction

Once the nutritional emergency is addressed and the GABA system is being supported, serotonin and dopamine reconstruction become the central biological targets. These systems were chronically disrupted by alcohol and are the substrate for the depression, anhedonia, and motivational deficits that drive relapse in alcohol recovery.

Supplement.	Dose.	Timing.	Alcohol-specific notes

5-HTP (precursor to serotonin).	50–200mg	Evening - 30 minutes before bed.	Serotonin is depleted by chronic alcohol use. 5-HTP provides the direct precursor. Start at 50mg — if tolerated, increase. Do not combine with SSRIs or SNRIs without physician guidance (serotonin syndrome risk). Do not take in the morning if mood elevation causes anxiety. Evening dosing supports serotonin conversion to melatonin for sleep.
L-Triptofano	500–2000mg	Good evening, taking a break from protein.	An alternative or complement to 5-HTP — slightly slower-acting as it requires the additional conversion step. May cause less overshoot than 5-HTP in sensitive individuals. Combined tryptophan + 5-HTP at lower doses of each is an option for those who find either alone too stimulating.
L-Tyrosine / NALT	500–1500mg	Good morning, no protein today.	Precursor de dopamina. Útil para la anhedonia y el déficit motivacional en la recuperación del alcohol, especialmente en perfiles "Warrior" de COMT o con deficiencia de DRD2, donde el apoyo a la dopamina es una prioridad. Comience después del día 7, cuando la fase aguda haya disminuido. No usar con inhibidores de la MAO.
SAMe	400–800mg	Good morning, haven't eaten yet.	Universal methyl donor — directly supports dopamine and serotonin synthesis and the methylation pathways that process them. Evidence for depression in alcohol recovery. CAUTION: contraindicated in bipolar disorder; can trigger mania. Check CYP interaction with any medications. Start low.
Rhodiola rosea	300–600mg	Morning.	Modulation of dopamine and serotonin; adaptogen; anti-fatigue. Mild antidepressant effect with a strong evidence

			base. Particularly useful during weeks 2–8 when motivation and energy are low, but the precursor stack alone is insufficient. Do not take late in the day—it's activating.

4.4 — Liver Support Protocol

THE CENTURION — The organ that processed every drop must now heal.

⚠ If you have known liver disease, cirrhosis, or significantly elevated LFTs, consult your physician before starting any supplement protocol. Some supplements are hepatotoxic in liver disease despite being safe in healthy liver function.

Supplement	Dose.	Timing.	Rôle spécifique au foie.
NAC (N-Acetyl Cysteine)	600–1200mg twice daily.	Morning and evening with food.	Glutathione precursor — NAC replenishes glutathione, the liver's primary antioxidant and detoxification molecule. Alcohol massively depletes hepatic glutathione. NAC is one of the most evidence-supported liver-protective supplements and simultaneously reduces alcohol craving through glutamate homeostasis. Dual-action essential.
Milk thistle (silymarin)	140–420mg standardisert (70–80 % silymarin).	With meals.	The most studied hepatoprotective botanical. Silymarin has anti-inflammatory, antioxidant, and antifibrotic effects in the liver — directly relevant to alcoholic liver disease at every stage except end-stage cirrhosis. Well-tolerated. One of the few supplements where the evidence in alcohol-induced liver disease is genuinely strong.
Alpha-lipoic acid (ALA)	300–600mg daily.	Take away from food for best absorption.	Both water and fat-soluble — reaches compartments that other antioxidants cannot. Regenerates glutathione, Vitamin C, and Vitamin E

			simultaneously. Reduces acetaldehyde toxicity. Particularly relevant for individuals with the ALDH2 variant who accumulate excess acetaldehyde.
TUDCA (tauroursodeoxycholic acid)	250–500mg daily.	With food.	A bile acid derivative with strong protective effects on the liver—it reduces stress on the endoplasmic reticulum and improves the function of mitochondria in liver cells. There's growing research into its use in treating alcoholic liver disease. It can be expensive, but it's worth considering for people with significant liver problems.
Fosfatidilcolina.	1–2g daily.	With food.	Essential phospholipid for liver cell membrane integrity. Alcohol disrupts hepatic phosphatidylcholine synthesis. Supplementation supports membrane reconstruction, bile flow, and overall hepatocyte function. Also provides choline for acetylcholine synthesis — relevant for cognitive recovery.

4.5 — The Craving Interrupt Protocol

THE CENTURION — Les envies de consommer de l'alcool ne sont pas des demandes. Ce sont des événements neurologiques.

Alcohol cravings in recovery operate through at least three distinct neurobiological channels — GABA deficit craving (anxiety-driven), dopamine deficit craving (reward-seeking), and opioid/endorphin deficit craving (social pain and longing). Understanding which channel is activating the craving changes how you respond to it.

Tipo di desiderio.	What it feels like.	Interrupción inmediata.
GABA-deficiency cravings (most common in alcohol recovery).	Anxiety-driven. The nervous system is sending an alarm signal. Alcohol quiets alarms. The craving is the nervous system requesting its most reliable alarm-silencer.	4-7-8 breathing immediately — activates the vagus nerve and provides measurable GABAergic relief. L-theanine 200mg. Cold water on face and wrists —

		activates the dive reflex, immediate parasympathetic activation. Magnesium if not already taken today.
Craving due to dopamine deficiency.	Flat, grey, joyless. The world has no colour. Activities that should feel good don't. The craving is the reward system requesting stimulation.	Intense physical activity – the quickest way to boost dopamine levels. Exposure to cold temperatures (e.g., a cold shower or ice bath). Positive social interaction with someone who makes you feel good. Do the most rewarding activity that comes naturally to you (see Section 6).
Craving for opioids/endorphins.	Lonely. Emotionally raw. Yearning for connection or comfort that feels out of reach. Often misread as craving for alcohol specifically when it is actually craving for the warmth and belonging alcohol's endorphin release was producing.	Contact with other people – physically if possible (a hug, a handshake), or at the very least, by voice. Do not isolate yourself when experiencing this craving. Exercise also releases endorphins. Laughter. Any activity that produces genuine oxytocin and endorphin co-release: playing with a pet, physical contact with someone safe, vigorous movement with another person.

THE 5-MINUTE CRAVING INTERRUPT — EXECUTE IN ORDER.

1. Anxiety-based.
2. EXECUTE THE 4-7-8 BREATH. Four full cycles. No exceptions. This is the universal first step regardless of craving type.
3. TAKE THE APPROPRIATE SUPPLEMENT. Magnesium + L-theanine for GABA craving. L-tyrosine or Rhodiola for dopamine craving (if it's morning and you haven't taken them). Neither replaces steps 1–2 — they support what your body is already doing.
4. La ubicación, lo que hay en su campo de visión o salir al aire libre pueden ayudar a romper el circuito del deseo.
5. MAINTENIR LE CONTACT. Un message ou un appel téléphonique. Pas pour parler de l'envie, mais simplement pour rester en contact. L'isolement est le terrain où les envies d'alcool se manifestent.
6. EXECUTE THE NATURAL REWARD. From Section 6. Whatever is highest-intensity and immediately available. You are practicing the redirect — not just resisting, but replacing.
7. Trigger, type, intensity (0–10), action, outcome. The documented craving becomes data. Data becomes leverage.

4.6 — Peptide Support Layer

The following peptides have mechanistic relevance to GABA system recovery, neuroinflammation reduction, and liver repair. Research compounds — physician discussion required.

Peptide.	Mechanism in alcohol recovery.	Timing.	Notes
BPC-157	Restauração do eixo intestino-cérebro; neuroplasticidade; modulação da dopamina e serotonina; ação anti-inflamatória; efeitos hepatoprotetores diretos em modelos de pesquisa.	Dag 1 en verder - dagelijks.	BPC-157 is the peptide with the greatest relevance in alcohol recovery because it simultaneously addresses three of alcohol's primary damage domains: gut barrier (leaky gut), dopamine-serotonin systems, and liver. Oral administration is appropriate and may be superior for gut healing. The repair of the gut-brain axis may be its most important function in alcohol recovery.
Selank	Modulation of GABA and serotonin; anxiolytic with no risk of dependence; reduces anxiety without causing drowsiness.	Weeks 1–8 — take as needed for anxiety; take daily if you're experiencing strong cravings for GABA.	Selank is particularly valuable in alcohol recovery because GABA-deficiency-related anxiety is the primary cause of cravings and the main risk factor for relapse. Selank addresses this issue without the addictive properties of benzodiazepines. It is non-sedating and non-habit-forming. Administered intranasally.
Epitalon	Sleep architecture; pineal pathway; telomere protection; circadian rhythm normalization.	Weeks 1–4 — 10-day cycle, then as needed.	Sleep disruption in alcohol recovery can persist for weeks to months. Epitalon supports the pineal pathway that alcohol chronically suppresses. It also provides cellular-level protection during the oxidative stress of early recovery.
Thymosin Beta-4 (TB-500)	Anti-inflamatorio; reparación de tejidos; reducción de la fibrosis hepática en modelos de	Months 1–3 if liver involvement is significant.	For those with alcoholic liver disease where fibrosis is present, TB-500 has documented anti-fibrotic effects in research models.

	investigación.		This is physician-territory given liver disease complexity. Not first-line but worth discussing with a physician managing your liver recovery.
Semax	BDNF upregulation; cognitive repair; prefrontal cortex support; dopamine and serotonin modulation.	Weeks 2 onwards.	Alcohol produces measurable cognitive impairment and prefrontal cortex thinning. Semax's BDNF-stimulating effects support the neural repair that is occurring during sobriety. Particularly valuable for the cognitive fog and motivational impairment of months 1–3.

Section 5 — Rebuilding Sleep

THE CENTURION — Alkohol hielp je niet om te slapen. Het maakte je alleen maar slaperig. Dat is niet hetzelfde.

One of the most common and most dangerous beliefs in alcohol use disorder is that alcohol helps sleep. Alcohol produces sedation — rapid loss of consciousness. It does not produce sleep. It suppresses REM sleep almost entirely, fragments sleep architecture, reduces slow-wave deep sleep, and produces rebound insomnia in the second half of the night as alcohol is metabolized.

The result is that years of "drinking to sleep" produce a brain that has never experienced genuine restorative sleep while drinking—and now, in early recovery, struggles to sleep at all because the sedation mechanism is gone and the brain has not practiced the natural sleep initiation process for years.

Rekonstruksi tidur dalam pemulihan alkohol adalah salah satu aspek terpenting dan paling diremehkan dari proses tersebut. Insomnia adalah penyebab utama yang sering disebut sebagai pemicu kekambuhan dini. Ini juga merupakan target yang sangat responsif terhadap suplemen dan protokol perilaku di bawah ini.

- Melatonin (0.5–1mg sublingual, 30–60 minutes before your desired bedtime) — a lower dose is often more effective than a higher dose; higher doses can disrupt your natural sleep-wake cycle.
- Magnesium glycinate (300–400mg, 1 hour before bed) — supports GABA, reduces glutamate excitotoxicity, lowers core temperature.
- Glycine (3–5g, 30 minutes before bed) — reduces core body temperature, the primary physiological sleep trigger; also an NMDA co-agonist that supports the GABA-glutamate rebalancing.
- L-Theanine (200–400mg evening) — promotes alpha waves, reduces anxious hyperarousal that prevents sleep onset.
- 5-HTP (50–100mg evening) — precursor to serotonin, which converts to melatonin; supports both mood and sleep.
- Fosfatidilserina (100-200 mg la sera) - riduce il cortisolo serale; la disfunzione dell'asse ipotalamo-ipofisi-surrene causata dal consumo cronico di alcol aumenta il cortisolo e impedisce l'inizio del sonno.

Protocol 17 (Sleep Architecture)

* **Alcohol Recovery Additions:** Avoid all alcohol, including cooking alcohol and mouthwash with alcohol. Even trace amounts can activate the reward circuit and disrupt sleep architecture in early recovery. The vivid dreams of early recovery are REM rebound – they are healing, not symptoms to suppress.

Section 6 — Porting the Addiction: Finding Your Own GABA.

THE CENTURION — Centurian Paper C-28 Applied to Alcohol Recovery.

Alcohol can induce at least three different neurochemical states depending on how, when, and why you use it. Each of these can be addressed with natural alternatives that won't leave you feeling depleted.

What Alcohol Was Actually Providing:

* **Euphoria**
* **Relaxation**
* **Reduced inhibitions**
* **Improved sociability**
* **Pain relief**
* **Anxiety reduction**
* **Sleep aid**

The function	The neurochemistry.	The natural replacement architecture.
Anxiety relief / nervous system quiet.	Potenziamento del GABA: l'alcol ha silenziato il sistema di allarme che non si fermava. Per la costituzione della persona "Sage" con livelli cronicamente elevati di sostanze chimiche legate allo stress, questo era il sollievo più efficace disponibile.	Breathwork (daily, non-negotiable). Meditation — the contemplative practice that builds the internal GABA system rather than borrowing against an external supply. Cold exposure (produces post-stress calm). Nature. Magnesium. The practice is daily and lifelong — not situational.
Social ease and connection.	Rilascio di endorfine + riduzione dell'inibizione sociale. L'alcol ha dissolto la barriera della autoconsapevolezza e ha prodotto calore, apertura e un senso di appartenenza.	The goal is not to become naturally as disinhibited as alcohol made you — it is to build genuine connection that does not require disinhibition. Twelve-step communities, small groups, sober social communities. Physical contact (safe and consensual) releases oxytocin. Service — helping others — produces endorphins

		and belonging simultaneously.
Plezier en beloning.	Attivazione dei sistemi dopaminergici e degli oppioidi. L'alcol fa sentire bene, soprattutto nelle prime fasi. Questo è reale. Si tratta di un vero evento neurochimico.	Multiple dopamine sources built deliberately: vigorous exercise, creative mastery, competitive activity, achievement with social stakes. No single source will replicate alcohol's full-system activation — which is why multiple sources are essential rather than optional.
Ritual de transición y permiso para descansar.	The drink after work wasn't mainly about pharmacology. It was about a signal – the day is over, you're allowed to relax, and you can stop working.	Same time, same location, different content. Non-alcoholic ritual drink (sparkling water, herbal tea, non-alcoholic cocktail). Brief ceremony of transition. The brain needs the signal; it does not need the alcohol in the signal.

The Natural GABA Architecture for Alcohol Recovery.

THE CENTURION — Building the Internal Brake System the Drug Was Running.

The Sage constitution's recovery—and alcohol recovery is predominantly, though not exclusively, a Sage story—requires building a life that provides sufficient nervous system calm through owned means. This is not about eliminating stimulation. It is about building a resilient internal regulation system that does not require a substance to function.

- Una práctica diaria de contemplación de al menos 10 minutos. No como una preferencia, sino como una necesidad biológica ineludible. Esto es lo que construye el tono endógeno de GABA que el alcohol estaba suplementando. El Protocolo 04 (Dharma Recovery) en el Compendio principal es la referencia.
- Vigorous aerobic exercise at least 4 times per week. The BDNF release from sustained aerobic exercise is the primary neural repair mechanism for alcohol-damaged brains. The endorphin release addresses the opioid/endorphin system simultaneously. This is not lifestyle advice — it is the most powerful non-pharmacological intervention for alcohol recovery that exists.
- A creative practice with genuine depth. The flow state is the natural GABA state — total absorption in a demanding activity quiets the anxious mind through the mechanism of full attentional engagement rather than chemical suppression. Writing, music, visual art, cooking, building. Depth over breadth.

- Authentic community. Not a performance-based community. Not a social media community. It involves in-person connection with people who understand your real situation, where it's okay to be yourself without pretense. The endorphin and oxytocin systems that alcohol triggers respond specifically to this type of interaction. Twelve-step communities offer this for many individuals—and the Compendium's Protocol 04 and Protocol 08 address the social aspects of recovery in general terms.
- Exposition à la nature comme intervention planifiée. Pas de manière fortuite. Un minimum de 20 minutes par jour. Les niveaux de cortisol du système nerveux en voie de guérison se normalisent de manière mesurable dans les environnements naturels, contrairement aux environnements urbains ou intérieurs.
- Service. The act of genuinely helping another person activates the reward, connection, and meaning circuits simultaneously and provides the sense of being useful in the world that alcohol's social lubricant was partially producing. Many people in long-term alcohol recovery report that service is the most reliable source of sustained wellbeing they have found.

Section 7 — Navigating the Recovery Kernel: Your Protocol Map.

Fase.	Protocols	Primary focus	Alcohol-specific notes
Medical Detox (Days 0–7 if physically dependent)	Medical provider — not the Compendium.	Survival. Seizure prevention. Medical stabilisation.	The Recovery Kernel framework doesn't start here. Medical detoxification begins here. Once you are medically stable, return to this guide and start at the Biological Foundation phase.
Biological Foundation (Days 1–14)	Protocol 07, Protocol 09, Protocol 17, Protocol 18.	Emergency nutrition, thiamine, magnesium, sleep, gut repair.	Thiamine on Day 1. Magnesium on Day 1. Full B-complex on Day 1. These are not optional. The depleted system cannot begin recovery without its raw materials. Protocol 18 (nutrition) and Protocol 09 (gut-brain axis) are the highest-priority Volume I protocols for alcohol recovery.
Genomic Mapping (Weeks 1–4)	Section 15 (Compendium), Protocol 11, Protocol 12	GABRA2 assessment, OPRM1 for naloxone guidance, COMT constitution, MTHFR.	The GABRA2 high-risk variant and OPRM1 A118G are the two highest-priority genomic findings in alcohol recovery — they directly determine medical treatment decisions (seizure monitoring, naltrexone appropriateness). Get tested as early as possible.
GABA System Repair (Weeks 1–12)	Protocol 04, Protocol 01 craving interrupt, Section 4.2 of this guide.	GABA supplement stack, breathwork, contemplative practice, anxiety architecture.	Unlike stimulant or cannabis recovery, GABA system repair is the PRIMARY biological target in alcohol recovery — it is both the most damaged system and the one whose deficit most directly drives relapse. Everything else happens within this container.
Behavioral and Environmental Architecture (Weeks 2–12)	Protocol 02, Protocol 05, Protocol 06, Protocol 08, Protocol 13	Prevenção da transferência de dependência, redesenho ambiental, mapeamento de gatilhos, arquitetura social.	Il trasferimento da alcol a benzodiazepine è il tipo di trasferimento di dipendenza più pericoloso dal punto di vista medico durante la riabilitazione. Il protocollo 02 deve essere letto immediatamente. Anche il trasferimento alla cannabis è comune tra i bevitori ansiosi. Elencare esplicitamente i rischi del

			trasferimento prima che si verifichino.
Liver and Physical Recovery (Months 1–6)	Protocol 07, Protocol 18, liver support stack (Section 4.4)	Liver function monitoring, NAC, silymarin, TUDCA, phosphatidylcholine.	Repeat LFTs at 1 month, 3 months, and 6 months of sobriety. The liver is one of the most regeneratively capable organs in the body — significant recovery is possible even from advanced damage with sustained abstinence. Track it. Celebrate the improvement.
Identity and Meaning (Months 2–12)	Protocol 20, Protocol 21, Protocol 23, C-28	Identity reconstruction, the sober self, purpose architecture.	Alcohol use disorder often deeply intertwines with identity — "I am someone who drinks." For long-term drinkers, sobriety requires constructing an identity that does not have a substance-shaped hole in the middle of it. Protocol 20 (identity reconstruction) is essential.
Long Game (Months 6–24+)	Protocol 24, Protocol 25, C-28, C-31	Purpose architecture, relational repair, the natural addiction fully operational.	La recuperación del volumen cerebral que ocurre en el segundo año de sobriedad es medible mediante resonancia magnética y genuinamente significativa. La persona que se comprometió con la recuperación en el mes 1 puede ser neurológicamente diferente, significativamente y mensurablemente mejor, para el mes 18. Esto no es una metáfora. Es el argumento biológico más poderoso a favor de la sobriedad a largo plazo.

Section 8 — The Medical Support Layer

L'alcolismo ha la più solida base di evidenza tra tutte le dipendenze per il trattamento farmacologico. I farmaci elencati di seguito sono sottoutilizzati: gli studi dimostrano costantemente che meno del 10% delle persone con alcolismo che potrebbero beneficiare di questi farmaci li riceve. Questo è un divario nel trattamento che costa vite umane.

Bawa bahagian ini kepada doktor anda. Tanya terus tentang pilihan-pilihan ini. Kerangka Kerja Pemulihan dan rawatan farmakologi tidak bersaing—ia direka untuk berfungsi bersama.

FDA-Approved Medications for Alcohol Use Disorder:

* **Naltrexone (ReVia, Vivitrol)**
* **Acamprosate (Campral)**
* **Disulfiram (Antabuse)**

Medication.	Mechanism.	Evidence level	Considerazioni genomiche e cliniche.
Naltrexone (oral or monthly injection / Vivitrol).	Opioid receptor antagonist — blocks the endorphin reward signal from alcohol, reducing the "wanting" and the reward.	Strong – supported by multiple randomised controlled trials; FDA approved.	OPRM1 A118G carriers show dramatically superior response — up to 3x better outcomes. If you know you carry this variant, naltrexone is likely your highest-efficacy medication. Monthly injection (Vivitrol) removes the daily compliance variable. The Sinclair Method — taking naltrexone before each drinking episode to extinguish the reward response — is a validated harm reduction approach for those not ready for abstinence.
Acamprosate (Campral)	GABA and glutamate modulation — reduces the excitatory overdrive of alcohol withdrawal and PAWS; reduces distress of abstinence.	Strong — Approved by the FDA; particularly effective for PAWS.	Particularly relevant for the GABA-deficit profile (GABRA2 high-risk) and for those with severe PAWS. Renally cleared — no liver metabolism, which is an advantage in alcohol recovery where liver function may be compromised. Does not interact with the liver

			support stack. Start during or immediately after detox.
Disulfiram (Antabuse)	Inhibition of ALDH2 — blocks acetaldehyde clearance, resulting in severe nausea, flushing, and discomfort if alcohol is consumed.	Moderate — aversion strategy; effective when compliance is supervised.	Works by making drinking immediately and severely punishing. Most effective when dosing is observed (partner, pharmacist, or program). Not appropriate for ALDH2*2 carriers — already have impaired acetaldehyde clearance; disulfiram would intensify an already existing biological deterrent. Requires absolute commitment to abstinence as it makes drinking medically dangerous.
GLP-1 agonists (semaglutide, tirzepatide)	Reduzir o desejo por álcool através da modulação dos circuitos de dopamina e recompensa; dados de 2025-2026 mostram uma redução de 50-60% nos episódios de consumo.	Emerging — strong observational data; clinical trials ongoing as of 2026.	The most significant development in alcohol treatment pharmacology in years. The mechanism appears to reduce the 'wanting' quality of alcohol craving — the compulsive pull toward the substance — rather than merely the reward once consumed. For patients with both AUD and metabolic concerns, this may address both simultaneously. Discuss with your physician.
Gabapentin.	GABA-modulating anticonvulsant — reduces withdrawal symptoms and PAWS; particularly relevant for sleep and anxiety.	Moderate — use outside approved indications; substantial evidence supports its use for alcohol use disorder (AUD).	Useful for managing PAWS (Post-Acute Withdrawal Syndrome) and sleep disturbances during the first 1–3 months. CAUTION: Gabapentin itself carries a risk of dependence, particularly in individuals with a history of addiction. Use should be limited in time and under physician supervision. Individuals with a GABRA2 high-risk profile may benefit most but also carry the highest risk of misuse — monitor carefully.

A Final Word.

THE CENTURION — The system that chose alcohol wasn't broken. It was solving a real problem badly.

The GABA system that could not quiet itself. The endorphin system that could not generate adequate social warmth. The dopamine system that could not find enough reward in the ordinary texture of daily life. The methylation system that could not synthesize sufficient serotonin. These are not character defects. They are biological vulnerabilities — specific, genetic, addressable.

L'alcol ha risolto questi problemi. Per anni, forse per decenni, li ha risolti. La soluzione era reale. E stava distruggendo tutto il resto nel processo.

What the Recovery Kernel offers is a different kind of solution — slower, less dramatic, requiring daily practice and biological support and community and time. But a solution that does not cost the things alcohol was costing. That does not narrow your life down to the single question of when the next drink will come.

The brain volume lost to alcohol grows back. The GABA receptors recover. The serotonin system rebuilds. The liver heals. The relationships that were strained can be repaired—not all of them, and not quickly, but meaningfully. The cognitive function that alcohol was impairing returns, often surpassing the pre-drinking baseline because the tools built in recovery—the breathwork, the sleep, the nutrition, the exercise, the contemplative practice—are actively building a more capable brain than the one that started drinking.

The Recovery Kernel was designed for individuals who required a more detailed guide than the standard one provided. You now have the alcohol-specific version of that guide.

The navigation continues in the main Compendium. Use it.

— — —

Refer to the Recovery Kernel Complete Centurion Compendium for all referenced protocols.
Crisis: call or text 988 | Emergency: 911 | SAMHSA: 1-800-662-4357